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INFLUENCE OF PECAN CULTIVAR AND SOURCE OF INOCULUM ON DEVELOPMENT OF PECAN SCAB

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Pecan scab, caused by *Cladosporium caryigenum* (Ell. et Lang.) Gottwald, is the most important disease of pecan (*Carya illinoensis* (Wang.)K.Koch) (Gottwald 1982). Immature shoots, twigs, leaves, and nut shucks are highly susceptible to infection by *C. caryigenum* (Gottwald 1985). Scab lesions are typically brown to black, olivaceous and velvety in appearance and severe infections can result in shoot and twig dieback .

Cladosporium caryigenum overwinters as subcuticular stromata in lesions on twigs, petioles, and nut shucks. Conidia produced in stromata in the spring are the primary inocula, and conidia produced in lesions throughout the growing season are the secondary inocula. Conidia are disseminated by wind and rain to susceptible young tissues. Conidia germinate and *C. caryigenum* infects host tissues subcuticularly when free moisture is present (Gottwald 1985, Latham and Rushing 1988). Though partial cultivar resistance to scab is available, control of this disease is primarily attained through the use of protectant and systemic fungicides (Ellis et al. 1998).

The long-term value of cultivar resistance to scab is limited by the ability of *C. caryigenum* to adapt to overcome that resistance. There is clear evidence for such pathogenic specialization in *C. caryigenum*. Different strains of the fungus appear to exist at different locations. A given cultivar may appear to be quite resistant at one location but may be found to be susceptible at another. Cultivars that were resistant to scab infection in the early days of the pecan industry, such as San Saba, Georgia, and Delmas, are now found to be scab susceptible (Cole 1957, Demaree and Cole 1929). The existence of different pathogenic strains on different cultivars and at different locations presents important considerations for the screening of pecan cultivars for scab resistance, as different strains may be present at different locations. Researchers testing the resistance of a given cultivar to scab should take care to include many sources for the *C. caryigenum* used in disease trials.

A clearer understanding of the pathogenic specialization of *C. caryigenum* would provide important information about the biology of this fungus and host resistance in pecan cultivars. Further information about pathogenic variation that occurs in *C. caryigenum* would be valuable in developing improved screening programs for cultivar resistance and the development of new scab-resistant pecan cultivars. Pathogenic variation in *C. caryigenum* was examined in this study by testing the effect of source of inoculum on infection of resistant and susceptible pecan cultivars. The study consisted of two experiments. The first experiment was a whole-plant inoculation study in which the interactive effects of source of inoculum and cultivar on symptom development were investigated. The second experiment was a detached leaf study in which the interactive effects of cultivar and source of inoculum on conidial germination, appressorium formation, and subcuticular fungal growth were compared.

In the first experiment, container-grown partially resistant (Sumner) and susceptible (Wichita) pecan cultivars were inoculated with conidia of *C. caryigenum* obtained from each cultivar. Leaf wetness was maintained on inoculated trees for 48 h at 25 C in growth chambers. Disease intensity was evaluated 17 days post inoculation, by visual estimation of disease severity (percent diseased tissue) and by counting the number of lesions per leaflet. The experiment was repeated for a total of three experimental trials. There was a significant effect of the cultivar-inoculum source on resulting disease intensity. The isolate of *C. caryigenum* from Wichita caused significantly less disease on Sumner trees than on Wichita trees. The isolate from Sumner caused significantly more disease on Sumner trees than did the isolate form Wichita. Apparently the isolate from Sumner had adapted to overcome the resistance of Sumner.

The presence of chlorotic halos in the resistant Sumner infections and the cross-inoculations of Wichita trees with inoculum from Sumner indicates that though these lesions were apparently smaller and less velvety in appearance, some disease reaction occurred. This kind of chlorotic halo is typical of the hypersensitive response that has been documented in other plant-pathogen interactions. It also indicates that physiological effects resulting from infection of *C. caryigenum* extend beyond the dark, central portion of the lesion where sporulation is evident. Converse

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(1960) reported similar chlorotic reactions in self and cross inoculations with four susceptible cultivars. Turechek (1995) found that lesions on leaves of the resistant cultivar Eliott and the moderately susceptible cultivar Cape Fear were similarly smaller and less velvety in appearance than lesions on the susceptible cultivar Wichita.

In the second experiment, detached leaves from Sumner and from Wichita were inoculated with conidia of *C. caryigenum* isolates obtained from each cultivar. Leaf wetness was maintained at 24 C for 96 h and leaf disks were sampled at 24, 48, 72, and 96 h after inoculation. Percentages of potential infection sites with germinated conidia, appressoria, and subcuticular growth were recorded. The cultivar-inoculum source interaction had no effect on an of these early infection events, up to and including subcuticular growth. The timing of these events was similar to the progression of these events in an earlier study of development of *C. caryigenum* on leaves of the susceptible pecan cultivar Schley (Latham and Rushing 1988). The percentage of each of these early stages of fungal development was not significantly different between 72 h and 96 h post inoculation.

This study has delimited a time period when the mechanisms of resistance and pathogenic variation are likely to be expressed. In the whole-plant experiment, there was a significant effect of the cultivar-inoculum source interaction on disease development. Significant differences were observed in disease intensity resulting from inoculations of Sumner pecans (resistant) and Wichita pecans (susceptible) with *C. caryigenum* isolated from each cultivar. At 72 h (3 days) post inoculation in the detached leaf experiment, there were no significant differences in the frequency of subcuticular growth between the cultivar-inoculum source combinations. Germination, appressorium formation, and subcuticular growth were not significantly different between 72 h (3 days) and 96 h (4 days) after inoculation. These results indicate that the observed differences in disease intensity in different cultivar-inoculum source combinations that appear in symptom development result from differences in fungal development between 96 h (4 days) and 17 days post inoculation. Since visible symptoms were first observed at 10 days post inoculation in each experimental trial, differences in infection resulting from specific cultivar-inoculum source combinations should be observed between 4 and 10 days post inoculation. These differences in fungal development may be manifested as differences in rate and extent of subcuticular growth.

This study provides clear evidence for pathogenic variation in *C. caryigenum* and delimits the time period where the resistant response of pecan cultivars to infection by *C. caryigenum* is likely to occur. This information about cultivar-specific pathogenic variation in *C. caryigenum* contributes to our basic understanding of varietal specialization in the imperfect fungi, an area in which there has been relatively little research. More research of pathogenic variation in *C. caryigenum* would also provide important information for pecan growers, since the long-term value of scab-resistant pecan cultivars is limited by the ability of the pathogen to overcome that resistance. Resistance screening for cultivar resistance to scab can be improved by using inoculum from multiple isolates of *C. caryigenum* in resistance trials. The observed differences in disease resulting from the specific cultivar-inoculum source combinations and the critical time period that has been delimited offer a unique opportunity to study pathogenic variation and resistance mechanisms in this economically devastating pathogen of pecan.

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